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Emergency Medicine Clinics North America: Blood Pressure Management in Neurologic Emergencies

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Introduction and Background

Blood pressure (BP) management is a cornerstone of acute care in neurologic emergencies, where timely and effective intervention is essential to reducing morbidity and mortality. Neurological complaints constitute about 5% of annual emergency department (ED) visits and are often associated with significant clinical urgency[1]. Elevated BP in these scenarios is not merely a secondary finding but often represents a modifiable factor with significant prognostic implications[2]. Emergency physicians frequently face pressure to act on elevated BP readings, even without signs of end-organ damage[3]. However, this review focuses on scenarios where prompt and precise BP control is essential—such as stroke, traumatic brain injury (TBI), preeclampsia, and other critical conditions—where early recognition and intervention may improve functional outcomes and lower mortality[4,5]. This article aims to provide clinicians with practical insights into current guidelines, emerging evidence, and ongoing controversies, offering a comprehensive, evidence-based resource to enhance patient outcomes in the complex landscape of neurologic emergencies. This review will begin with a brief overview of hypertension in the emergency department, followed by a focused discussion on the evidence and blood pressure management strategies for specific neurologic conditions.

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Approach to Undifferentiated Hypertension

Hypertension affects nearly 1.3 billion people globally, yet only about 20% have adequate BP control[6]. In the United States, elevated BP is observed in up to 45% of emergency department (ED) visits[7], but hypertensive emergencies—marked by acute end-organ damage—are diagnosed in only about 0.6% of cases[6]. According to ACEP clinical policy[8], routine testing or initiation of antihypertensive medications for isolated elevated BP without symptoms is not recommended.

The primary role of emergency physicians is to identify hypertensive emergencies through detailed history, vital sign trends, and focused physical exams assessing cardiovascular, neurological, ophthalmologic, and renal involvement. Testing should be limited to cases with suspected end-organ damage, using tools such as ECG, metabolic panels, chest radiographs, urinalysis, or brain imaging as needed.

For patients with severe BP elevation but no acute symptoms, management should consider comorbidities, chronicity of BP elevation, follow-up access, and social determinants of health[9]. Prescribing antihypertensive medication at discharge may be reasonable if follow-up is delayed[10]. Ultimately, a patient-centered approach, focusing on distinguishing between transient or chronic BP elevations and true emergencies, helps reduce the long-term burden of uncontrolled hypertension while addressing acute risks appropriately[11].

Undifferentiated Stroke

Background

The optimal approach to blood pressure management in patients with suspected stroke prior to advanced imaging remains uncertain, as high-quality evidence guiding prehospital and early emergency care is limited. While extensive research has informed in-hospital BP management for confirmed ischemic and hemorrhagic strokes, evidence for hyper-acute BP management in suspected strokes is limited[12]. Elevated BP in ischemic stroke may act as a compensatory mechanism to maintain cerebral perfusion in the ischemic penumbra, rapid or excessive BP reduction could theoretically compromise perfusion and exacerbate ischemic injury. In contrast, elevated BP in intracerebral hemorrhage (ICH) can contribute to hematoma expansion, making BP control a potentially beneficial intervention.

For patients eligible for intravenous thrombolysis (IVT), achieving a BP target of ≤185/110 mmHg is critical to minimize the risk of hemorrhagic transformation. This target is based on guidelines from the American Heart Association/American Stroke Association (AHA/ASA) and the European Stroke (ESO).[13,14] However, in the absence of imaging confirmation, the benefits and risks of prehospital BP lowering remain unclear. Effective BP management is an accessible and low-cost intervention, making it a compelling target for improving early stroke outcomes, yet the absence of definitive evidence complicates decision-making in the prehospital and hyper-acute settings.

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Key Trials

- PIL-FAST:[15] A small UK feasibility study testing oral lisinopril in suspected stroke patients with systolic BP >160 mmHg. The treatment group had lower BP at hospital arrival and 4 hours post-admission, but the small sample size (14 patients) and lack of follow-up limited its conclusions.
- RIGHT and RIGHT-2:16–17 In RIGHT (41 patients), transdermal GTN reduced BP by 18 mmHg and improved 90-day outcomes. RIGHT-2 (1,149 patients) found no functional or mortality benefit, suggesting feasibility but uncertain effectiveness.
- INTERACT-4:[18] This Chinese trial (2,404 patients) compared intensive BP lowering (130–140 mmHg) vs. usual care. Outcomes improved in hemorrhagic stroke but worsened in ischemic stroke, underscoring challenges in pre-imaging BP management.

Management

These trials underscore the complexity of prehospital BP management, with the type of stroke playing a critical role in determining the risks and benefits of early intervention. The European Stroke Organisation, reflecting on these findings, currently advises against routine BP lowering in the prehospital setting due to the potential risks associated with misclassifying stroke types.[14]

Lowering BP in suspected stroke before imaging is not currently recommended by international guidelines. However, careful monitoring of BP trends in both prehospital and hospital settings remain an essential component of stroke care. While hypertensive emergencies may warrant BP control upon hospital arrival, this should be balanced with imaging prioritization in accordance with established guidelines. Proactively planning for BP management during imaging and ensuring clear communication with nursing staff and pharmacy, when available, can help facilitate timely and coordinated BP control when necessary.

Conclusion

BP management in suspected stroke prior to advanced imaging must be individualized based on the patient's clinical presentation and transport context. While achieving a systolic BP <185 mmHg and diastolic BP <110 mmHg is critical for patients being considered for thrombolysis, overly aggressive BP reduction in undifferentiated strokes remains controversial. This balanced approach minimizes potential harm while maximizing the likelihood of favorable outcomes.

Acute Ischemic Stroke

Background

Acute ischemic stroke (AIS) accounts for approximately 87% of all strokes, with up to 75% of patients presenting with elevated BP on admission.[19–20] While BP elevation may

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help maintain cerebral perfusion, excessive hypertension is associated with poor outcomes, including hemorrhagic transformation and increased mortality.[21] The approach to BP management varies based on eligibility for intravenous thrombolysis (IVT) or endovascular therapy (EVT), requiring a balance between ischemic risk and hemorrhagic complications.

Blood Pressure Management Based on Reperfusion Eligibility

Non-Reperfusion Candidates: For AIS patients not receiving IVT or EVT, the optimal BP target remains uncertain. Observational studies suggest a U-shaped relationship, where both very high and very low BP correlate with worse outcomes.[22–24] AHA/ASA and ESO guidelines recommend withholding antihypertensive therapy unless SBP >220 mmHg or DBP >120 mmHg, except for specific comorbidities.[13–14] Aggressive BP reduction can impair cerebral perfusion, particularly in large vessel occlusions with impaired autoregulation.

Key Trials

- SCAST:[25] Randomized 2,029 patients with AIS or ICH to candesartan vs. placebo within 30 hours. BP reduction was modest, with no effect on functional outcomes, mortality, or recurrence.
- PRoFESS:[26] Telmisartan vs. placebo in 1,360 AIS patients within 72 hours. BP differences were minor, with no impact on outcomes.[27]
- ENOS:[29] Transdermal GTN vs. placebo in 4,011 patients within 48 hours. BP was lower at 24 hours, but there was no sustained functional benefit at 90 days.

Clinical implication:

These trials show no consistent benefit of routine BP lowering in AIS patients not undergoing reperfusion therapy. At the bedside, consider BP reduction only for severe hypertension (SBP >220 mmHg or DBP >120 mmHg) or for other compelling indications.

Thrombolysis Candidates

IVT with alteplase (tPA) or tenecteplase (TNK) is a cornerstone of AIS treatment but increases the risk of intracerebral hemorrhage (ICH), especially with uncontrolled hypertension.[30] AHA/ASA and ESO guidelines recommend lowering BP to ≤185/110 mmHg before IVT and maintaining ≤180/105 mmHg for 24 hours post-thrombolysis.[13–14] These recommendations are partly based on the NINDS tPA trial which excluded patients with BP >185/110 mmHg, which was supported by pilot and observational studies. [31–34]

Key Trials

- SITS Registry:[35] Observational data found a fourfold increased risk of symptomatic ICH in patients with SBP >170 mmHg compared to those within the 141–150 mmHg range.

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- ENCHANTED:[36] Intensive BP lowering (target SBP 130–140 mmHg) reduced ICH rates but did not improve 90-day functional outcomes, supporting current guidelines of SBP <185/110 mmHg pre-IVT and <180/105 mmHg post-IVT.

Clinical implication:

These findings reinforce strict BP control before and after IVT to minimize hemorrhagic complications. However, aggressive BP lowering beyond guideline thresholds has not demonstrated functional benefit and may risk cerebral hypoperfusion.

Endovascular Therapy Candidates

BP management for EVT remains an area of active investigation. Elevated BP before, during, and after EVT is associated with an increased risk of hemorrhagic transformation, while excessive BP lowering may impair collateral circulation and infarct salvage.[37] Based on international guideline, BP should be maintained ≤180/105 mmHg during and for 24 hours post-EVT, though evidence supporting this threshold is based primarily on early trials where most EVT patients also received IVT.[13–14,38] However, high quality observational data elevated systolic and/or diastolic BP (>220/120 mmHg) correlates with worse outcomes, including symptomatic ICH and mortality. [39–40]

Key Trial: BP-TARGET:[41] Compared post-EVT BP targets of 100–129 mmHg vs. 130–185 mmHg, finding no significant difference in ICH rates, though adherence to intensive BP goals was low.

Clinical Implication:

A BP goal of ≤180/105 mmHg remains reasonable post-EVT, pending further evidence. While extremely elevated BP (>220/120 mmHg) should be treated, overly aggressive reduction may be harmful.

Pharmacologic Management

Intravenous (IV) antihypertensive agents are preferred in acute ischemic stroke due to their rapid onset and titratability. The choice of agent depends on the patient's clinical status, comorbidities, and BP response. Please refer to Box 1 for reasonable medications with dose ranges.

Box 1:

Intravenous Antihypertensives in Neurologic Emergencies

Agent	Mechanism of Action	Recommended Use
Nicardipine	Calcium channel blocker	First-line agent; initiate at 5 mg/h IV, titrate by 2.5 mg/h every 5–15 min as needed (max 15 mg/h).[42]
Labetalol	β-blocker with α-blocking effects	Initial bolus: 10–20 mg IV over 1–2 min; may repeat every 10 min with increasing doses (20–80 mg), total cumulative dose not to exceed 300 mg. Alternatively, initiate continuous infusion at 2–10 mg/min following bolus.[42]

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Agent	Mechanism of Action	Recommended Use
Clevidipine	Short-acting calcium channel blocker	Initiate at 1–2 mg/h IV, titrate by doubling the dose every 2 min as needed (max 21 mg/h).[42]
Hydralazine	Direct vasodilator	Administer 5–10 mg IV over 1–2 min; may repeat as needed (max cumulative dose: 20 mg). Use cautiously due to unpredictable hypotensive response.[42]
Esmolol	Ultra-short-acting β-blocker	Loading dose: 50–500 mcg/kg IV over 1 min, followed by continuous infusion at 25–50 mcg/kg/min. Titrate by 20–50 mcg/kg/min every 5 min as needed (max 300 mcg/kg/min).[42]
Nitroprusside	Arterial and venous vasodilator	Initial: 0.3 to 0.5 mcg/kg/minute; titrate every 5 minutes to desired effect; usual dose: 3 mcg/kg/minute; maximum dose: 10 mcg/kg/minute.[42] Avoid unless absolutely necessary (risk of increased ICP).[43]

BP should be monitored every 5–15 minutes initially, with adjustments based on patient response. Neurological checks should be performed frequently to ensure BP reduction is not compromising cerebral perfusion.

Conclusion

BP management in ischemic stroke remains a nuanced clinical decision requiring individualized treatment based on reperfusion eligibility. While the ideal BP target continues to be debated, current evidence supports moderate BP control to prevent hemorrhagic complications while maintaining cerebral perfusion. Ongoing trials will further refine optimal BP thresholds for both thrombolysis and endovascular therapy, ultimately improving outcomes in ischemic stroke patients.

Intracerebral Hemorrhage

Background

Intracerebral hemorrhage (ICH), sometimes referred to as hemorrhagic stroke, accounts for ~10% of all strokes and remains the most lethal subtype, with mortality rates up to 40% despite advances in acute stroke care.[44–45] Elevated BP at presentation is a major modifiable factor, strongly linked to hematoma expansion, neurologic deterioration, and worse functional outcomes.[46–47] The pathophysiology of BP elevation in ICH is multifactorial, driven by sympathetic activation, pain, stress, and chronic hypertension.[48] While anticoagulant use has contributed to rising ICH incidence, the shift to direct oral anticoagulants (DOAC) over vitamin K antagonists (VKA) may help mitigate this trend.[49–50] Given that most hematoma expansion occurs within the first 3 hours, timely BP reduction is a critical target in acute ICH management.[51] However, clinical trials have yielded neutral or mixed results, leaving uncertainty about optimal BP targets and treatment timing.

Key Trials

- INTERACT:[52] In 404 ICH patients, intensive BP lowering (SBP <140 mmHg) significantly reduced hematoma growth without increasing adverse events

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- INTERACT-2:[53] Among 2,839 patients, intensive BP reduction showed no difference in death or major disability, but ordinal analysis suggested modest functional benefit. Safety was comparable between groups.
- ATACH-II:[54] Randomized 1,000 patients to SBP 110–140 mmHg vs. 140–180 mmHg using nicardipine. No functional benefit, but increased renal adverse events with intensive therapy.
- INTERACT-3:[55] An international trial testing a care bundle including BP lowering to <140 mmHg within 1 hour. Functional outcomes improved, but the specific contribution of BP control is unclear due to similar BP levels across groups.
- INTERACT-4:[18] Prehospital BP reduction (target 130–140 mmHg) improved outcomes in ICH patients but worsened outcomes in ischemic stroke, highlighting the risk of treating before stroke subtype is confirmed.

Clinical Implication:

Early BP reduction to <140 mmHg in ICH is likely safe and may reduce hematoma expansion, though functional outcome benefits are modest. Caution is warranted when treating suspected stroke pre-imaging, as aggressive BP lowering may harm ischemic stroke patients.

BP Targets and Guidelines for ICH

Given the mixed trial results, expert guidelines provide practical recommendations:

AHA/ASA 2022 Guidelines:[56]

- In patients with spontaneous ICH requiring acute BP lowering, careful titration to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes.
- In patients with spontaneous ICH in whom acute BP lowering is considered, initiating treatment within 2 hours of ICH onset and reaching target within 1 hour can be beneficial to reduce the risk of hematoma expansion and improve functional outcome.
- In patients with a mild-to-moderate ICH with SBP ≥ 150 mg Hg, lowering to 130–150 mmHg is reasonable and safe.
- In patients with spontaneous ICH presenting with large or severe ICH or those requiring surgical decompression, the safety and efficacy of intensive BP lowering are not well established.

ESO 2021 Guidelines:[14]

- In patients with acute (<24 hours) intracerebral hemorrhage there is continued uncertainty over the benefits and risks (advantages/disadvantages) of intensive blood pressure lowering on functional outcome.

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- Hyperacute ICH (<6 hours): Recommend lowering BP <140 mmHg but keeping it >110 mmHg to minimize hematoma expansion.
- Extended BP Management: Maintain BP control for at least 24 hours and up to 72 hours to reduce ongoing bleeding risk.

Pharmacologic Management

IV antihypertensives are preferred due to rapid onset and titratability, ensuring smooth, sustained BP control to avoid large fluctuations, which are associated with worse outcomes.[57] Please see ox 1 for antihypertensive recommendations.

Conclusion

Blood pressure management in ICH is both time-sensitive and clinically critical. Rapid but controlled BP reduction within the first 2–3 hours can limit hematoma expansion, with current evidence supporting a target systolic BP of 130–150 mmHg in most cases. While this strategy appears safe and potentially beneficial, ongoing research aims to further define optimal thresholds and tailor BP management to individual patient profiles.

Transient Ischemic Attack: Management at Discharge

Patients with suspected transient ischemic attack (TIA) who are deemed low risk, are often discharged from the emergency department (ED) with neurology follow-up after an initial work-up. While the risk of recurrence remains high, it has declined with improved secondary prevention.[58–59] RCTs show that prescribing antihypertensives reduces recurrent TIA and stroke risk.[60] Ideally, initiation should be coordinated with outpatient, but prescribing at discharge may be appropriate if follow-up delays are expected.

AHA/ASA guidelines and other national recommendations advise continuing, restarting, or initiating antihypertensives after TIA, targeting BP <130/80 in most cases.[42,61–62] Thiazide diuretics, ACE inhibitors, and ARBs are first-line options supported by large trials76–79 and CCBs are reasonable alternatives if needed.[63–65]

Antihypertensive therapy should be individualized based on comorbidities. Please refer to Box 2 outlining recommended starting and maximum doses for commonly prescribed agents.

Box 2:

Standard Oral Antihypertensives

Agent	Mechanism of Action	Starting Dose / Max Dose	Considerations
Hydrochlorothiazide	Diuretic	12.5–25 mg once daily/50 mg daily[42]	First-line for patients without comorbidities
Lisinopril	ACE Inhibitor (ACEi)	5–10 mg once daily/40 mg daily[42]	Preferred for patients with diabetes and proteinuria

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Agent	Mechanism of Action	Starting Dose / Max Dose	Considerations
Losartan	Angiotensin II Receptor Blocker (ARB)	25–50 mg once daily/100 mg daily[42]	Suitable for patients intolerant to ACE inhibitors

Conclusion

For low-risk TIA patients discharged from the ED, antihypertensive therapy plays a key role in secondary prevention. When timely outpatient follow-up is uncertain, initiating treatment at discharge is reasonable. Targeting a BP <130/80 mmHg is recommended, using agents tailored to comorbid conditions. Thiazide diuretics, ACE inhibitors, and ARBs remain first-line options.

Posterior Reversible Encephalopathy Syndrome

Background

Posterior reversible encephalopathy syndrome (PRES) is a neurologic disorder with acute to subacute onset, presenting with encephalopathy (~80%),headache (~50%), seizures (~75%), visual disturbances (~60%), and focal deficits (5–15%).[66–69] First described in 1996 as reversible posterior leukoencephalopathy syndrome, it is characterized by vasogenic edema, typically in the parieto-occipital regions on MRI.[68,70–71] Though hypertension is a common feature (~70%), it is absent in up to 20% of cases.[66] PRES remains understudied, with most data derived from case series and meta-analyses.[67]

PRES affects all ages but is most common in women aged 30–50 years.[70–71] It is associated with hypertension, renal disease, sepsis, immunosuppressive therapy, autoimmune disorders, and preeclampsia.[67,71–74]. Two theories explain its pathophysiology: the hyperperfusion theory, which attributes it to BP fluctuations causing vascular leakage;[66–67] and the toxic theory, which links endothelial dysfunction from endogenous or exogenous toxins, particularly in immunosuppressed and septic patients.[75]

Pharmacologic Management

BP control is the cornerstone of treatment, though no trials have assessed its direct impact on outcomes.[73,76] Consensus amongst experts reducing SBP by ≤25% in the first hour, with normalization over 24–48 hours, while avoiding excessive BP fluctuations.[67] First-line antihypertensives include clevidipine, labetalol, and nicardipine (see box 1). Early identification and management of underlying triggers, such as infection, medications, or autoimmune disorders, are essential.[73,77] Worrisome medications include, but not limited to, immunosuppressive agents, chemotherapy agents, monoclonal antibodies, bone marrow stimulating agents, and high dose steroids. [77] Seizures should be treated with anticonvulsants, but prophylaxis is not recommended.[73] Magnesium should be maintained within a normal range due to its neuroprotective effects.[73] Given the risk of status epilepticus and intracranial hemorrhage as well as frequent use of IV antihypertensives, ICU monitoring is often warranted.

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Conclusion

PRES requires early recognition, BP control, and management of underlying triggers. Careful hemodynamic monitoring and targeted therapy can improve outcomes and prevent complications.

Preeclampsia and Eclampsia

Background

Hypertensive disorders of pregnancy are among the leading cause of maternal and perinatal mortality worldwide, responsible for approximately 16% of maternal deaths.[78] Preeclampsia is defined as new-onset hypertension (systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg) after 20 weeks gestation accompanied by proteinuria or signs of end-organ dysfunction.[78] Eclampsia refers to the onset of seizures in women with preeclampsia. These conditions present after 20 weeks gestation, during labor, or within six weeks postpartum, though onset before 34 weeks is uncommon.[79]

Emergency physicians should promptly evaluate BP ≥140/90 mmHg in pregnant or postpartum patients and urgently treat severe hypertension (≥160/110 mmHg) to prevent complications such as stroke, heart failure, and myocardial ischemia.[78] Preeclampsia is diagnosed by sustained hypertension plus proteinuria or end-organ dysfunction (severe features).[78] Risk factors include antiphospholipid syndrome, assisted reproduction, obesity (BMI >30), chronic hypertension, diabetes, kidney disease, age ≥35, multifetal gestation, OSA, lupus, prior preeclampsia, and thrombophilia.[80] Though exam findings may be minimal, new-onset edema, petechiae, or RUQ tenderness warrant further evaluation.[78] Please refer to Box 3 for preeclampsia diagnostic criteria, modified from the American College of Obstetrics and Gynecology (AGOG) practice guideline.[78]

Box 3:

Diagnostic Criteria for Preeclampsia *(Asterisk indicates severe features)*

	Criteria
Blood Pressure	- SBP ≥140 mmHg or DBP ≥90 mmHg on two occasions, ≥4 hours apart after 20 weeks gestation in a patient with previously normal BP OR - SBP ≥160 mmHg or DBP ≥110 mmHg (can confirm within minutes to expedite treatment)*
AND	**Proteinuria**
	- ≥300 mg per 24-hour urine collection - Protein/creatinine ratio ≥0.3 mg/dL - Dipstick reading of 2+ (only if other methods unavailable)
OR*	**In the absence of proteinuria, new-onset hypertension with the new onset of any of the following:**
	- Thrombocytopenia: Platelet < 100,000/μL - Renal insufficiency: Serum creatinine >1.1 mg/dL or doubling from baseline (without other renal disease) - Hepatic Dysfunction: Liver transaminases ≥2 times normal - Pulmonary edema - Neurologic symptoms: Persistent headache unresponsive to medication (excluding alternative diagnoses), visual disturbances

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Pharmacologic Management

Emergent antihypertensive therapy is indicated for severe preeclampsia (SBP ≥160 mmHg or DBP ≥110 mmHg, or with severe features), ideally within 30 minutes of confirmation.[78] For preeclampsia without severe features, BP management should be promptly discussed with obstetrics. Magnesium sulfate administration is essential for seizure prophylaxis in severe preeclampsia.

Please refer to Box 4 which are recommended antihypertensive regimens, in accordance if AGOG guidelines with no significant efficacy differences among choices.[78]

Box 4:

Antihypertensive Regimens in Preeclampsia/Eclampsia

Agent	Dose	Notes
Labetalol	10–20 mg IV initially; then 20–80 mg every 10–30 min (max 300 mg) or continuous infusion 1–2 mg/min IV	Avoid with asthma, cardiac dysfunction, or bradycardia
Hydralazine	5–10 mg IV every 20–40 min (max cumulative dose 20 mg), or infusion 0.5–10 mg/hr	May cause maternal hypotension or abnormal fetal heart tracings
Nifedipine (immediate release)	10–20 mg orally; repeat in 20 min if needed, then every 2–6 hours; max daily dose 180 mg	May cause reflex tachycardia

Conclusion

Effective management of preeclampsia and eclampsia in the ED emphasizes prompt diagnosis, controlled BP reduction, seizure prophylaxis, obstetrics consultation, and vigilant monitoring to improve maternal and fetal outcomes.

Reversible Cerebral Vasoconstriction Syndrome

Background

Reversible cerebral vasoconstriction syndrome (RCVS) is a rare, underrecognized cause of thunderclap headache, marked by transient cerebral artery narrowing.[81] It can cause stroke, TIA, hemorrhage, and PRES due to impaired autoregulation.[82] Triggers include serotonergic drugs, vasoconstrictors, stimulants (e.g., cocaine, methamphetamines), cannabis, postpartum state, and physical or emotional stress.[82] RCVS predominantly affects women (2–10:1 ratio), with onset around age 45.[83] Angiography often reveals a classic “sausage-on-a-string” pattern.[84] Blood pressure management is important in RCVS, as both hypo- and hypertension may worsen outcomes.[81] About 25–30% of patients experience BP surges during their course.[85] While no standard BP target exists, keeping SBP <160 mmHg appears reasonable, given the higher hemorrhagic risk above this threshold.

Pharmacologic Management

Calcium channel blockers (CCBs) like nimodipine (30–60 mg q4h) and extended-release verapamil (120 mg q12–24h) are used to relieve vasospasm in RCVS.[81] However, they have not shown consistent benefit in outcomes and may increase ischemic risk if BP drops too

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low.[85, 86] A reasonable approach is to titrate BP based on symptoms—aiming to reduce thunderclap headache severity while avoiding hypotension—and maintain SBP below 160 mmHg to minimize the risk of hemorrhage without compromising cerebral perfusion.

Conclusion

RCVS is a complex cerebrovascular condition characterized by reversible arterial narrowing and significant blood pressure fluctuations, requiring careful management. While calcium channel blockers are commonly used, individualized blood pressure control remains critical to minimize complications and improve outcomes. Further research is needed to establish optimal treatment strategies.

Traumatic Brain Injury

Background

Traumatic brain injury (TBI) is a major source of morbidity, mortality, and disability, affecting an estimated 2.9 million people annually in the U.S., with over 60,000 deaths and 5 million living with long-term deficits.[87] TBI severity ranges from concussion to coma. Blood pressure management is critical, as both hypotension and hypertension worsen outcomes.[88]

Following TBI, cerebral autoregulation is impaired, making cerebral blood flow (CBF) highly dependent on maintaining an optimal cerebral perfusion pressure (CPP), which is determined by mean arterial pressure (MAP) minus intracranial pressure (ICP).[89] Hypotension reduces cerebral perfusion pressure (CPP = MAP – ICP), risking ischemia, hypoxia, and neuronal death—strongly linked to increased mortality.[90] Conversely, hypertension can raise intracranial pressure (ICP) and cerebral edema, also worsening prognosis.[91]

Optimal Blood Pressure Targets

The 2017 Brain Trauma Foundation guidelines recommend maintaining SBP >100 mmHg for patients aged 50–69, and >110 mmHg for those 15–49 or over 70, though evidence was insufficient for strong endorsement.[92] Recent studies suggest no single threshold, but rather a BP range associated with reduced mortality.[93] The 2023 3rd Edition Prehospital Guidelines emphasize avoiding hypotension and recognizing that both low and high BP can worsen outcomes, a principle extendable to ED care.[94]

Key Studies:

- Chestnut RM, et al:[95] A prospectively collected data set from the Traumatic Coma Data Bank demonstrated that a single episode of hypotension was associated with at two-fold increase in mortality.
- EPIC TBI study (secondary analysis):[96] In over 12,000 prehospital TBI cases, mortality rose sharply with SBP <90 mmHg, but risk increased across the 40–

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119 mmHg range. The lowest adjusted mortality was observed with SBP 130–180 mmHg.

- IMPACT Study:[97] A retrospective cohort examining the relationship of thresholds for SBP and MAP demonstrated SBP ranges from 120–150 mmHg and MAP ranges from 85–110 mmHg are thresholds to target improved outcomes.

Clinical Implication:

Maintaining SBP ≥110–120 mmHg, and ideally in the 130–150 mmHg range, is associated with better outcomes in TBI. Hypotension—even brief or mild—can double mortality, and high-quality evidence supports shifting prehospital and in-hospital targets upward from the traditional 90 mmHg threshold. Frequent BP monitoring and proactive correction are essential in minimizing secondary brain injury.

Pharmacologic Management

Volume resuscitation is essential in trauma care to maintain perfusion and oxygen delivery to the brain. Hypotension should be corrected promptly with isotonic fluids or blood products.[94] Hypertonic fluids, such as hypertonic saline or mannitol, may be used in severe TBI (GCS <8) when elevated ICP is suspected.[94] No fluid type has shown superiority in outcomes, and albumin is contraindicated due to increased mortality.[98]Vasopressors like norepinephrine and phenylephrine are used to maintain CPP, though data comparing agents are limited.[99] Outcomes appear similar between agents, so selection should be guided by clinical context (e.g., sepsis, neurogenic shock).[100]

Conclusion

Blood pressure management in TBI is a delicate balance—both hypotension and hypertension can worsen brain injury and negatively affect outcomes. Guidelines recommend maintaining SBP >100 mmHg for patients aged 50–69 and >110 mmHg for others, with emerging evidence supporting a target range of SBP 110–150 mmHg. Preventing and promptly correcting hypotension while avoiding excessive hypertension is crucial to preserve cerebral perfusion and reduce secondary brain injury. Tailored resuscitation strategies using fluids and vasopressors should be guided by patient-specific factors. Further research is needed to refine precise BP targets and optimize treatment strategies in this complex population.

Conclusion

Optimal blood pressure management in neurologic emergencies requires a nuanced, patient-centered approach, balancing the risk of cerebral hypoperfusion against complications associated with excessive hypertension. While current evidence and guidelines inform BP targets across various neurologic conditions—including ischemic stroke, intracerebral hemorrhage, preeclampsia/eclampsia, PRES, RCVS, and traumatic brain injury—significant

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controversies and research gaps persist. Emergency clinicians must carefully integrate clinical presentation, underlying pathophysiology, evolving literature, and expert recommendations to individualize BP management strategies. Future randomized controlled trials will be essential to refine these guidelines further, enhancing patient outcomes through precise, evidence-based interventions tailored to each neurologic emergency.

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Key Points

- Blood pressure management is crucial in neurologic emergencies to reduce morbidity and mortality.
- Elevated blood pressure in neurologic emergencies can be a modifiable factor with significant prognostic implications.
- Emergency physicians play a key role in managing blood pressure in conditions like stroke, RCVS, PRES, TBI, and preeclampsia.
- The review provides a comprehensive framework for managing blood pressure in various neurologic emergencies, including stroke and traumatic brain injury.
- Current guidelines, emerging evidence, and ongoing controversies in blood pressure management are discussed to improve patient outcomes.

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Synopsis

- The document “Blood Pressure Management in Neurologic Emergencies” from Emergency Medicine Clinics North America emphasizes the importance of timely and effective blood pressure management in neurologic emergencies. It highlights that elevated blood pressure is a significant prognostic factor in conditions such as stroke, traumatic brain injury, PRES and preeclampsia. The review provides a detailed examination of current recommendations and controversies, offering a practical resource for emergency physicians. This article aims to equip clinicians with the knowledge to balance rapid intervention with long-term risk mitigation, ultimately improving patient outcomes.

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Clinics Care Points

- Differentiate hypertensive emergencies from incidental hypertension: treat only when there is acute end-organ damage or compelling indication; isolated ED hypertension does not require immediate reduction.
- Stroke care hinges on timing and subtype: in acute ischemic stroke, maintain BP ≤185/110 mmHg for thrombolysis/EVT; in intracerebral hemorrhage, early smooth lowering to 130–150 mmHg reduces hematoma expansion.
- Avoid overcorrection: aggressive or rapid BP reduction can worsen cerebral ischemia (AIS, RCVS, PRES) or cause renal injury (ICH); target gradual, controlled lowering.
- Pregnancy-related hypertension is unique: severe preeclampsia/eclampsia (≥160/110 mmHg) requires treatment within 30 minutes with labetalol, hydralazine, or nifedipine, plus magnesium for seizure prophylaxis.
- Traumatic brain injury demands prevention of hypotension: maintain SBP >100–110 mmHg depending on age; both hypotension and severe hypertension are linked to increased mortality.